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Characterization of the detailed interaction interface between T. brucei telomere proteins TRF and TIF2

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Abstract

The protozoan parasite *Trypanosoma brucei* causes fatal African trypanosomiasis in humans and nagana in cattle. *T. brucei* switches its variant surface glycoproteins (VSGs) inside the mammalian host, evading the host immune response. VSGs are expressed monoallelically from subtelomeric expression sites, and telomere proteins regulate VSGs.

We previously found that telomere protein *Tb*TIF2 interacts with *Tb*TRF (TTAGGG-repeat binding factor) and plays important roles in VSG switching regulation. *Tb*TRF maintains the telomere terminal structure. *Tb*TIF2 is essential for subtelomeric integrity and suppresses VSG switching by inhibiting subtelomeric gene conversion. Depletion of *Tb*TIF2 decreases *Tb*TRF protein level. We hypothesize that *Tb*TRF-*Tb*TIF2 interaction is essential for maintaining *Tb*TRF protein level. We test this hypothesis by mapping the interaction between *Tb*TRF and*Tb*TIF2. *Tb*TRF has both N-terminal TRF Homology (TRFH) and C-terminal Myb domains. *Tb*TRFH contains seven helices and interacts with the N-terminus of *Tb*TIF2 (aa 2-190). We found that deleting either of the first two helices in *Tb*TRFH abolishes its interaction with *Tb*TIF2. Currently we are generating deletion and point mutations within the first two helices of *Tb*TRFH, which will be tested for their ability to interact with *Tb*TIF2 to determine the key residues in *Tb*TRFH that are required for interacting with TbTIF2.

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